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A Study Quantifying the Hand-to-Face Contact Rate and Its Potential Application to Predicting Respiratory Tract Infection

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A substantial portion of human respiratory tract infection is thought to be transmitted via contaminated hand contact with the mouth, eyes, and/or nostrils. Thus, a key risk factor for infection transmission should be the rate of hand contact with these areas termed target facial membranes. A study was conducted in which 10 subjects were each videotaped for 3 hr while performing office-type work in isolation from other persons. The number of contacts to the eyes, nostrils, and lips was scored during subsequent viewing of the tapes. The total contacts per subject had sample mean $\bar{x} = 47$ and sample standard deviation $s = 34$. The average total contact rate per hour was 15.7. The authors developed a relatively simple algebraic model for estimating the dose of pathogens transferred to target facial membranes during a defined exposure period. The model considers the rate of pathogen transfer to the hands via contact with contaminated environmental surfaces, and the rate of pathogen loss from the hands due to pathogen die-off and transfer from the hands to environmental surfaces and to target facial membranes during touching. The estimation of infection risk due to this dose also is discussed. A hypothetical but plausible example involving influenza A virus transmission is presented to illustrate the model.

Keywords hand-to-face contact, infection transmission, pathogen exposure model

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INTRODUCTION

It is thought that a substantial portion of human respiratory tract infections are transmitted via contaminated hand contact with the mouth, eyes, and/or nostrils, with subsequent transport to target tissue sites in the oro- and nasopharyngeal region. Aside from rhinovirus infection, which has been shown to occur following contact of the nasal and conjunctival mucosa with fingertips seeded with virus,[1] the evidence for the hand contact route is indirect albeit substantial. Intervention studies conducted in senior daycare facilities,[2] student dormitories,[3] military barracks,[4] and households[5] have shown that increased handwashing and/or hand treatment with an iodine solution decreased respiratory tract illnesses by 20% or more relative to the incidence in control groups. A meta-analysis of eight selected intervention studies geared toward the general public showed a 24% decrease in respiratory illness relative to control groups due to handwashing measures.[6] Given these overall findings, recent pandemic influenza planning documents identify hand contact as a potential exposure route,[7] even though this pathway never has been experimentally demonstrated for seasonal influenza A virus.

The risk of respiratory tract infection due to hand touches to target facial membranes (the conjunctivae of the eyes, the lips, the mucous membranes of the nostrils) depends, in part, on the rate of contact (number per unit time) with these targets. Other risk factors include the rate of hand contact with environmental surfaces, the degree of contamination of the touched surfaces, the transfer efficiency on touching, and the infectivity (virulence) of the pathogen. A reasonable qualitative statement is that infection risk increases as the rate of hand contact with target facial membranes increases. However, a more quantitative description is desirable. Because there are sparse published data concerning the rate of hand contact with target facial membranes, the authors conducted a limited observational study of that rate. The authors also developed a mathematical framework that uses hand contact rate information to estimate the pathogen dose to target facial membranes and relates this dose to infection risk.

METHODS

The authors conducted an observational study into which 10 student volunteers (5 women and 5 men) were recruited. Each subject sat alone at the same desk in the same small room, and performed office-type work (e.g., working on a laptop computer, reading, writing) for a continuous 3-hr period while being videotaped, with the subject's knowledge. The subjects understood that: (i) the tapes would be viewed by the

investigators to count the number of touches to a variety of surfaces; and (ii) the tapes would be treated as confidential and kept in a locked cabinet in the office of one of the investigators (MN). Each subject was paid a small amount for participating. Subsequent to the 10 sessions, the same investigator (DB) viewed the tapes and counted the number of times each subject touched his/her eyes, nostrils, and lips. Descriptive statistics (the sample mean and standard deviation) were computed for the touch frequencies. Spearman rank correlation coefficients were computed for the three pairwise combinations of touch frequencies (lips-eyes, lips-nostrils, eyes-nostrils), and tests of the hypothesis that the rank correlation coefficients exceeded zero were performed.[8] The study design was reviewed and approved by the Committee for the Protection of Human Subjects, University of California, Berkeley.

Development of the mathematical model for the transfer of pathogens to target facial membranes was based on traditional principles of mass (or number) balance and the assumption of first-order loss rates. A traditional one-hit dose-response function was slightly modified to relate the risk of infection to the pathogen dose delivered to target facial membranes.

RESULTS OF THE OBSERVATIONAL STUDY

The data collected were the number of contacts of the three target facial membranes by each of 10 subjects over a 3-hr period while the subjects worked alone at a desk.

Table I lists the frequency of contacts with the eyes, nostrils and lips, and the sum of these counts. For the total counts per subject, the sample mean $\bar{x} = 47$, the sample standard deviation $s = 35$, and the sample range is 3 to 104. The average total contact rate per hour is 15.7 (the average total frequency divided by 3). There is substantial interindividual variability in total hand contact rates with target facial membranes, as reflected by the 35-fold difference in the range limits ($104 \div 3$) and by the 73% coefficient of variation, equal to $100\% \times (35 \div 47)$. There is also a positive intra-subject correlation between the number of hand contacts with lips and eyes and with lips and nostrils, which is to say that those who touch their lips frequently tend to touch their eyes and nostrils frequently.

TABLE I. Numbers of Hand Contacts Observed During a Continuous 3-Hour Period

Subject	Eyes	Lips	Nostrils	Total
1	0	0	3	3
2	4	2	1	7
3	2	12	4	18
4	1	1	20	22
5	10	22	15	47
6	13	33	8	54
7	17	15	27	59
8	6	31	28	65
9	9	52	30	91
10	12	72	20	104
$\bar{x}$	7.4	24	16	47
s	5.7	24	11	35

The Spearman rank correlation coefficients for contacts between different facial target sites are as follows: (i) 0.76 for the lips and eyes; (ii) 0.66 for the lips and nostrils; and (iii) 0.44 for the eyes and nostrils. Given the conventional hypothesis test error of alpha equal 0.05, the lips-eyes correlation coefficient of 0.76 is significantly greater than zero (one-sided p-value $= 0.011$), the lips-nostrils correlation coefficient of 0.66 is significantly greater than zero (one-sided p-value $= 0.024$), but the eyes-nostrils correlation coefficient of 0.44 is not significantly greater than zero (one-sided p-value $= 0.093$).

THE EXPOSURE MODEL

A model is presented that incorporates the hand contact rate and other factors that logically should influence the number of pathogens transferred to target facial membranes. It is advised that absent systematic experimental investigation, this exposure model must be viewed as providing a relatively crude first-pass estimate of the pathogen dose to target facial membranes.

For a given pathogen of interest, let $C_{surface}$ (pathogens per cm^2) denote the average viable pathogen density on those environmental surfaces that can be touched by a person; substantial variability in pathogen density at different surface locations is expected. Note that in the microbial risk literature, a small inanimate object (for example, a cup or a doorknob) that can carry pathogens and transfer them to the hands upon contact is termed a "fomite." Because broad surfaces such as tabletops are not considered fomites but can transfer pathogens to the hands, the authors prefer to use the term "environmental surface." The average environmental surface area touched per hand contact is denoted $A_{surface}$ (cm^2 per contact). A fraction f_{12} of the pathogens on the touched surface area are transferred to the hand. The subscript 12 denotes transfer from the environmental surface (1) to the hands (2). The rate of hand contact with environmental surfaces is $H_{surface}$ (contacts per minute). The rate of viable pathogens transfer to the hands (number of pathogens per minute) is:

$$\text{Rate of Transfer to the Hands} = H_{surface} \times C_{surface} \times A_{surface} \times f_{12} \quad \textbf{(1)}$$

At the same time, viable pathogens are lost from the hands due to die-off (inactivation), transfer back to touched surfaces, and transfer to target facial membranes. Pathogen die-off on the hands is reasonably modeled as exponential with a first-order rate constant α_{dieoff} (fraction per minute),[9] such that the fraction of pathogens that die in a short time interval Δt is $\alpha_{dieoff} \times \Delta t$. Let C_{hand} (pathogens per cm^2) denote the viable pathogen density on the hands. Let A_{hand} (cm^2) denote the contaminated hand surface area. Thus, the rate of viable pathogen die-off on the hands (number of pathogens per

minute) is:

$$\text{Rate of Die-Off on the Hands} = C_{hand} \times A_{hand} \times \alpha_{dieoff} \quad \textbf{(2)}$$

The rate of viable pathogen transfer back to environmental surfaces (number of pathogens per minute) is the product of the rate of hand contact with environmental surfaces $H_{surface}$, the viable pathogen density on the hands C_{hand}, the hand surface area involved in a touch, and the fraction f_{21} of the pathogens on the hand surface that are transferred to the environmental surface. The subscript 21 denotes transfer from the hand (2) to the environmental surface (1). For simplicity, the authors assume that the same hand area always touches environmental surfaces, such that the hand surface area involved in a touch is the previous parameter A_{hand}. Note that this equality is not a required assumption. The transfer rate back to environmental surfaces is:

$$\text{Rate of Transfer to Surfaces} = H_{surface} \times C_{hand} \times A_{hand} \times f_{21} \quad \textbf{(3)}$$

The rate of viable pathogen transfer to target facial membranes (number of pathogens per minute) is the product of the rate of hand contact with target facial membranes H_{face} (contacts per minute), the viable pathogen density on the hands C_{hand}, the hand surface area A_{hand} involved in a touch, and the fraction f_{23} of the pathogens on the hand surface that are transferred to the target membrane. The subscript 23 denotes transfer from the hand (2) to the target facial membranes (3). The transfer rate to target facial membranes is:

$$\text{Rate of Pathogen Transfer to Target Membranes} = H_{face} \times C_{hand} \times A_{hand} \times f_{23} \quad \textbf{(4)}$$

In terms of the rate of change of the viable pathogen number on the contaminated hand surface, Eq. 1 is a gain term and Eqs. 2–4 are loss terms. The rate of gain minus the rate of loss leads to the following differential equation for the number of viable pathogens on the hand surface:

$$A_{hand} \times \frac{dC_{hand}}{dt} = H_{surface} \times C_{surface} \times A_{surface} \times f_{12} - C_{hand} \times A_{hand} \times \alpha_{dieoff} - H_{surface} \times C_{hand} \times A_{hand} \times f_{21} - H_{face} \times C_{hand} \times A_{hand} \times f_{23} \quad \textbf{(5)}$$

For mathematical simplicity, the authors assume that the contaminated hand surface area A_{hand} is equal to $A_{surface}$, the environmental surface area touched per hand contact. In this case, dividing both sides of Eq. 5 by A_{hand} cancels all the surface area terms. Note that equality between A_{hand} and $A_{surface}$ is not a required assumption. Next, if the factors $C_{surface}$, $H_{surface}$, H_{face}, α_{dieoff}, f_{12}, f_{21} and f_{23} are treated as constants, then C_{hand} is the only unknown term. For $C_{hand} = 0$ at time zero, the solution equation for C_{hand} as a function of time t (minutes) is as follows:

$$C_{hand}(t) = \frac{H_{surface} \times C_{surfcace} \times f_{12}}{\alpha_{dieoff} + H_{surface} \times f_{21} + H_{face} \times f_{23}} [1 - \exp(-[\alpha_{dieoff} + H_{surface} \times f_{21} + H_{face} \times f_{23}] \times t)] \quad \textbf{(6)}$$

where f_{12}, f_{21} and f_{23} are fractions transferred per contact. For notational simplicity, let $\lambda_{decay} = \alpha_{dieoff} + H_{surface} \times f_{21} + H_{face} \times f_{23}$, in which case Eq. 6 is written as:

$$C_{hand}(t) = \frac{H_{surface} \times C_{surfcace} \times f_{12}}{\lambda_{decay}} \times [1 - \exp(-\lambda_{decay} \times t)]. \quad \textbf{(7)}$$

Next, consider that a person occupies a room or physical space with pathogen-contaminated surfaces for a continuous period of T minutes. The mean concentration of C_{hand} over the interval [0, T] is:

$$\overline{C_{hand,T}} = \frac{1}{T}\int_0^T C_{hand}(t)\,dt = \frac{H_{surface} \times C_{surface} \times f_{12}}{T \times \lambda_{decay}}\left[T + \frac{\exp(-\lambda_{decay} \times T) - 1}{\lambda_{decay}}\right] \quad \textbf{(8)}$$

The expected dose D_T of viable pathogens transferred to target facial membranes over the interval [0, T] is:

$$D_T = H_{face} \times A_{surface} \times \overline{C_{hand,T}} \times f_{23} \times T \quad \textbf{(9)}$$

In Eq. 9, if exposure duration T is much greater than the time scale for the pathogen loss rate from the hands (or $T \gg 1/\lambda_{decay}$), the steady-state solution to Eq. 7, equal to the quotient $(H_{surface} \times C_{surface} \times A_{surface} \times f_{12}) \div \lambda_{decay}$, can be substituted for the time-averaged quantity $\overline{C_{hand,T}}$. Subsequent to time T when no viable pathogens are being added to the hands, the concentration $C_{hand}(T)$ will not suddenly go to zero (unless the hands are cleaned in some manner) but will exponentially decrease with the loss rate constant λ_{decay}. In turn, there will be an additional dose to target facial membranes during this decay phase. If the interval subsequent to time T is defined as $[0, T_{decay}]$, the mean concentration of C_{hand} over the interval is:

$$\overline{C_{hand,Tdecay}} = \frac{C_{hand}(T)}{T_{decay} \times \lambda_{decay}}[1 - \exp(-\lambda_{decay} \times T_{decay})] \quad \textbf{(10)}$$

In turn, the expected dose D_{Tdecay} of viable pathogens transferred to target facial membranes over the interval $[0, T_{decay}]$ is:

$$D_{Tdecay} = H_{face} \times A_{surface} \times \overline{C_{hand,Tdecay}} \times f_{23} \times T_{decay} \quad \textbf{(11)}$$

The expected total dose D_{total} is the sum of D_T and D_{Tdecay}. This construct is a simplified version of a more complex exposure model that accounts for variability in the value of $C_{surface}$ due to pathogen additions to the surface (e.g., from settled cough particles) and pathogen losses from the surface involving die-off, transfer to the hands, and possible suspension into air.[10] The more complex model also considers different types of environmental surfaces with different contact rates, and incorporates room ventilation information such that the inhaled dose of respirable pathogens can be estimated.

THE INFECTION RISK FUNCTION

The authors use a one-parameter exponential model that assumes that a single pathogen can infect the host with a

probability denoted α. Let D_{tissue} denote the expected number of pathogens that deposit at target tissue sites in the oro- and nasopharynx. If each pathogen acts independently to initiate infection, the risk of infection R is:[11]

$$R = 1–\exp(-\alpha \times D_{tissue}) \quad \textbf{(12)}$$

This model can account for variable host susceptibility by treating α as variable across individuals, but the authors do not account for that circumstance because their emphasis is on exposure assessment and not the functional form of the risk equation. Note that the one-parameter exponential model is consistent with observed dose-infection response data for a variety of pathogenic viruses.[11] The parameter α is related to the infectious dose 50% (ID_{50}) value by the expression: $\alpha = \ln(2) \div ID_{50}$, where $ID_{50} \geq \ln(2)$.

The parameter α is pathogen specific and may vary substantially across target tissue sites within the respiratory tract. For example, depending on the pathogen, α might be much greater for deposition in the pulmonary region than for deposition in the pharyngeal region due to the density of receptor sites or host defense mechanisms. Evidence for such site-specific differences would be the observation that to achieve infection of 50% of test subjects (or animals), inhalation of far fewer pathogens is required if the pathogens are carried on respirable particles compared with inspirable but nonrespirable particles. This circumstance must be considered because few if any pathogens deposited on target facial membranes will penetrate into the respiratory tract past the epiglottis; thus, using an α estimate based on a respirable pathogen inhalation study may be inappropriate. A better study for estimating α for a pathogen received by the hand contact route would involve instillation of the pathogen directly onto oro- and nasopharyngeal membranes.

The latter idea segues to another consideration — the fraction of D_{total}, the expected total number of pathogens depositing on target facial membranes, that reach oro- and nasopharyngeal target sites. Does $D_{tissue} = D_{total}$, or does $D_{tissue} = \varepsilon \times D_{total}$, where $0 < \varepsilon < 1$? It is reasonable to believe that $\varepsilon < 1$, but the authors have seen no pertinent data. Therefore, perhaps the most appropriate study of infectivity for a pathogen received by the hand contact route would involve seeding the pathogen onto a target facial membrane, because infection response would inherently account for the fraction transported to oro- and nasopharyngeal target sites and for the α value at those sites. The estimated α value from such a study could be used in the equation: $R = 1 - \exp(-\alpha \times D_{total})$, where D_{total} is the expected total dose to the target facial membranes.

A HYPOTHETICAL EXAMPLE FOR EXPOSURE/RISK ASSESSMENT

Consider exposure to influenza A virus in a residential bedroom due to attending a sick family member (the infector). The duration of continuous exposure is $T = 30$ min, with a subsequent decay interval of $T_{decay} = 30$ min. Posit that $C_{surface} = 28$ $TCID_{50}$ cm^{-2}. The unit $TCID_{50}$ denotes the tissue culture infectious dose 50%, which is an operational quantity designating an unknown number of virus particles observed to infect 50% of replicate cell cultures each receiving the same volume of virus inoculum. It is likely that a $TCID_{50}$ unit corresponds to more than one virus particle. The $C_{surface}$ value is derived as follows.

Virus-containing particles are emitted in coughs. Assume the infector coughs 12 times per hour (0.2 cough min^{-1}), which is the approximate 40th percentile of the cough rates seen in pneumonia patients.[12] Emitted cough particles range in diameter from less than 1 μm to greater than 2,000 μm, but more than 99% of the aerosol volume (and presumably the emitted virus) is in large particles with diameters greater than 100 μm.[13] These large particles tend to settle rapidly onto room surfaces close to the point of emission. An estimated 0.044 mL of fluid (saliva) is emitted per cough.[13] Peak concentrations of influenza A virus in nasal fluid among a small panel of subjects were found to range from 6×10^2 to 2×10^7 $TCID_{50}$ mL^{-1}.[14]

Consider a plausible concentration in saliva to be 1×10^6 $TCID_{50}$ mL^{-1}. Thus, the assumed virus deposition rate onto room surfaces is (0.2 cough min^{-1}) $\times$ (0.044 mL $cough^{-1}$)$\times$ (1×10^6 $TCID_{50}$ mL^{-1}) = 8.8×10^3 $TCID_{50}$ min^{-1}. If the infector is quiescent, virus is removed from room surfaces primarily by die-off due to environmental stress. Estimated first-order die-off rate constants for an influenza A strain were 1.6×10^{-2} min^{-1} on pajamas and 2.0×10^{-3} min^{-1} on stainless steel.[15] Assume a plausible value of 1.0×10^{-2} min^{-1}. Consider the area of particle settling to be a circle with radius 1 m around the infector, such that the contaminated surface area is 3.1×10^4 cm^2. Thus, the average steady-state (constant) value $C_{surface}$ = (8.8×10^3 $TCID_{50}$ min^{-1}) $\div$ [(1.0×10^{-2} min^{-1})(3.1×10^4 cm^2)] = 28 $TCID_{50}$ cm^{-2}.

The hand contact rate with environmental surfaces depends on the individual and the activities being performed, but a plausible value is $H_{surface} = 1$ min^{-1}. The die-off rate on the hands appears to range from 4×10^{-1} $min^{-1}$to 2×10^{-2} min^{-1}.[15] Assume that $\alpha_{dieoff} = 1 \times 10^{-1}$ min^{-1}. The hand contact rate with target facial membranes also depends on the individual, but posit $H_{face} = 0.8$ min^{-1}, which corresponds to the average total contact rate in Table I. The environmental surface area touched by the hand per contact, and the surface area of the hand that does the touching, are also variable and need not be equal. For simplicity, posit that $A_{surface} = 2$ cm^2, which is the approximate area of a finger tip.

Virus transfer efficiency from a nonporous surface to a fingertip has been estimated to be 0.5% per touch per fingertip.[16,17] The authors could not locate published data on virus transfer efficiency from a porous surface to a fingertip, but the transfer efficiency for bacteria from a porous surface has been estimated to be 0.1% per touch per fingertip.[16] For room surfaces in general, the authors posited that $f_{12} = 3 \times 10^{-3}$(0.3%). Due to lack of data on microbial transfer efficiency from the hands to environmental surfaces, they assumed that $f_{21} = f_{12}$. The transfer efficiency of virus from a fingertip to the

lips has been estimated to be 35% per touch.[18] It was assumed the same value for the eyes and nostrils such that $f_{23} = 3.5 \times 10^{-1}$.

At this point, all the input factors for computing $\overline{C_{hand,T}}$ and $C_{hand}(T)$ have been specified and are summarized in Table II. Based on Eqs. 5 and 6, respectively, $C_{hand}(T) = 0.22$ $TCID_{50}$ cm^{-2} and $\overline{C_{hand,T}} = 0.20$ $TCID_{50}$ cm^{-2}. Based on Eq. 8, $\overline{C_{hand,Tdecay}} = 0.019$ $TCID_{50}$ cm^{-2}. Based on Eqs. 7 and 8, respectively, $D_T = 3.4$ $TCID_{50}$ and $D_{Tdecay} = 0.32$ $TCID_{50}$, such that the expected total dose D_{total} is 3.7 $TCID_{50}$. If there are multiple 30-min exposure periods followed by 30-min decay periods, and if the inputs remain the same across these periods, the total expected dose would be the corresponding multiple of 3.7 $TCID_{50}$.

Next, let $\varepsilon = 0.5$ (the midpoint of the 0 to 1 range), such that $D_{tissue} = 1.85$ $TCID_{50}$. Influenza A dose-infection response data from a nasal instillation study with human volunteers[19] was analyzed by the authors to estimate α. That analysis (to be reported elsewhere) yielded $\alpha = 5.7 \times 10^{-5}$ per $TCID_{50}$. Note that this estimate is subject to substantial uncertainty, because the pre-exposure antibody titers of the human subjects were unknown, and antibody titers are inversely related to the probability of developing clinical influenza. However, given $\alpha = 5.7 \times 10^{-5}$ per $TCID_{50}$, the estimated infection risk due to hand contact for a 30-min exposure period followed by a 30-min decay period is:

$$R = 1 - \exp(-\alpha \times D_{tissue})$$
$$= 1 - \exp(-5.7 \times 10^{-5} \times 1.85) = 0.00011$$

An influenza infection risk of 0.011% does not seem substantial. On the other hand, there is substantial uncertainty in the value of α for influenza A virus. If its value were 1000-fold greater than assumed here and if the D_{tissue} value were unchanged, infection risk would be 10%. In addition, multiple exposure periods would increase the cumulative infection risk.

DISCUSSION

This investigation is apparently one of only two studies on the rate of hand contact with target facial membranes. In a 1973 study by Hendley et al.,[1] the investigators observed a total of 124 adults seated either in an amphitheater or a Sunday school for periods of 30 to 50 min each, such that there were 89 person-hours of observation; in contrast, the present study involved 30 person-hours of observation. The Hendley study reported 29 episodes of nose-picking (0.33 hr^{-1}) and 33 episodes of eye-rubbing (0.37 hr^{-1}); the degree and duration of contact that qualified as nose-picking and eye-rubbing were not defined. These statistics are far lower than the rates are reported in Table I for, respectively, contacts with the nostrils (5.3 hr^{-1}) and the eyes (2.5 hr^{-1}).

TABLE II. Hand Contact Exposure Model Inputs for the Hypothetical Example Involving a 30-Minute Visit to an Infector's Bedroom

$C_{surface} = 28$ $TCID_{50}$ cm^{-2}
$H_{surface} = 1$ min^{-1}
$H_{face} = 0.8$ min^{-1}
$\alpha_{dieoff} = 0.1$ min^{-1}
$A_{surface} = 2$ cm^2
$f_{12} = 3 \times 10^{-3}$
$f_{21} = 3 \times 10^{-3}$
$f_{23} = 0.35$

Moreover, at least 50% of the nostril and eye touches that were observed could be classified as, respectively, nose-picking and eye-rubbing, although the authors rely on qualitative judgment for that classification. Assuming there was a true difference in the rates of nose-picking and eye-rubbing observed in the Hendley study and the present study, one reason might be that the present study subjects were alone and, thus, did not feel as socially inhibited as they would in a group setting. Beyond the issue of what constitutes nose-picking and eye-rubbing, the authors believe it is better to use the total contact rate with one or more target facial membranes to make an exposure estimate because pathogens could be transferred by both light and vigorous contact.

A future refinement in exposure estimation might be to consider the rates of light vs. vigorous contacts, with a unique transfer efficiency associated with each type of contact. It is also reasonable to speculate that the contact rate depends on the type of activity being performed during the exposure period. For example, an activity requiring manual handling of objects might limit hand contact with the face.

As previously stated, the exposure model must be viewed as providing a relatively crude first-pass estimate of the pathogen dose to target facial membranes. At the same time, the model incorporates factors that logically should influence pathogen transfer to target facial membranes, and identifies key information needs. In brief, quantitative data are needed concerning value ranges for: (i) pathogen concentrations on room surfaces; (ii) the rate of contact with potentially contaminated room surfaces; (iii) transfer efficiencies upon contact; and (iv) pathogen die-off rates on the hands. Information on pathogen die-off rates on environmental surfaces has recently been reviewed by Boone and Gerba.[9]

With respect to infection risk, the authors do not assert that Eq. 12 is the single best risk model for all pathogens, although it is a reasonable construct to apply when the dose-response data are sparse. An equally important issue is the value of the transport efficiency parameter ε. It is plausible that ε could be close to zero or close to one depending on the tissue receptor sites for the pathogen and the target facial membrane. For example, if pathogen tissue receptors were primarily in the nasopharynx above the soft palate, it seems likely that few if any pathogens deposited on the lips would reach those receptors. On the other hand, if the tissue receptors were primarily in the oropharynx below the soft palate, perhaps the great majority of pathogens deposited on the lips would reach receptor sites.

The field of quantitative microbial risk assessment is far less developed than that of toxic chemical risk assessment. Methods for determining the microbial quality of water and food have existed for decades, but descriptions of potential pathogen exposure have primarily been of a binary nature, that is, exposed vs. not exposed. Quantitative exposure assessments have been applied more recently to pathogens in water and food, but the hand contact exposure route has not been systematically investigated. It is hoped that the authors' observational data and hand contact exposure modeling will contribute to the future development of more rigorous microbial risk assessment tools.

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